

Letters

RESEARCH LETTER

Effects of a Chemical Sunscreen on UV-Induced Changes of Different Histological Features in Melanocytic Nevus

Several studies have investigated the effect of UV radiation on melanocytic nevi by using different approaches and different sources and types of irradiation.¹ We read with interest the work of Carrera et al,² and we would like to describe our experience also.³

Methods | In 2003 we selected 26 melanocytic nevi from 26 patients (male to female ratio, 12:14; mean age, 31.0 years; median age, 31.5 years [range, 21.0-62.0 years]) for a study approved by the local institutional review board of our university (application No. 11-024 ex 00/01). A sunscreen with sun protection factor (SPF) of 6.2 (containing UV-A and UV-B filter)⁴ was applied exactly to one-half of each nevus by using a tape to avoid contamination of the other half. Twenty minutes after the application of the sunscreen, the whole nevus was irradiated with 3 individual minimal erythema doses (MEDs) (dose range, 8.6-25.0 J/cm²) of solar-simulated UV radiation produced by an Oriel 1000 W Xenon light source (Oriel Corp), equipped with filters as previously described.⁴ Clinical and dermoscopic images were acquired using a digital camera (Nikon Coolpix 4000) equipped with a polarized dermatoscope (DermLite Foto; 3 Gen) at baseline (day 0) before sunscreen application and UV irradiation and at day 3 and day 7 when the nevus was excised. The skin adjacent to the unprotected half was labeled by a 1-mm color ink spot. For each nevus, a transversal section was reviewed by 1 of us (C.M.) in a blinded fashion to record the following criteria in both halves (protected and unprotected): thickness of the epidermis, pigmentation of the basal layer, dilated vessels in the papillary dermis, dilated vessels in the reticular dermis, melanocytes in the upper epidermis, atypical melanocytes (in all epidermal layers), dendritic melanocytes in the epidermis, and sunburn cells. In addition, sections immunohistologically stained with LCA, S100, MIB-1, and HMB-45 antibodies were analyzed. Statistical analysis was performed with a GraphPad Prism (version 4.0).

Results | Dermoscopy at day 3 showed an increase of erythema and a more pronounced pigment network in the unprotected halves but without statistical differences ($P > .05$) compared with the protected halves. At day 7 we observed an increase of brown to black globuli, brown dots, bluish white veil, atypical network, and increased vessels in both protected and unprotected halves without statistical differences between the 2 halves ($P > .05$).

Statistical analysis also did not show any differences between the 2 halves concerning the histopathological criteria

described herein, apart from a trend ($P = .06$) for more atypical melanocytes in all epidermal layers in the unprotected half of the same nevus.

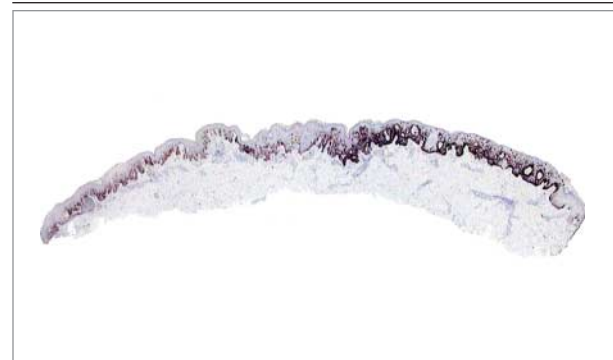
The HMB-45 stain resulted in significantly stronger staining in the unprotected halves compared with the protected ones (Wilcoxon signed rank test; $P = .02$) (Figure).

Discussion | Our experience is similar to the findings of the study conducted by Carrera et al,² with differences residing in the sources (we used an Oriel 1000 W Xenon, which emitted a more relevant UV spectrum than that used by Carrera et al²) and doses of irradiation (we used 3 MEDs solar-simulator UV irradiation and Carrera et al² used 2 MEDs of UV-B) and SPF.²

Apart from the fact that regression was not observed by us, our dermoscopic findings are similar to those of Carrera et al² and were unexpected also for us, especially in regard to our previous experience.⁵ In contrast, Manganoni et al⁶ found an increase in size and changes in dermoscopic features, including overall darkening, increased pigment network expression, formation of branched streaks, and increased number and size of brown globules and dots in unprotected nevi compared with no changes in sunscreen-protected nevi. It should be emphasized that Manganoni et al⁶ irradiated the melanocytic nevi with narrowband UV-B or UV-A1.

Regarding the histopathological criteria, apart from a trend ($P = .06$) for more atypical melanocytes in all epidermal layers in the unprotected half of the same nevus, in our experience statistical analysis did not show any differences between the 2 halves. Carrera et al² observed statistically significant differences between unprotected and protected halves for parakeratotic hyperkeratosis, marked lentiginous melanocytic hyperplasia, suprabasal solitary melanocytes, and prominent and elongated melanocyte dendrites and assumed that these changes were UV induced because observed only in the unprotected half.²

Figure. HMB-45 Antibodies Stain



The HMB-45 antibodies stain was significantly stronger in the unprotected half (right side, dark blue margin) compared with the protected half.

In contrast to the findings of Carrera et al,² in our study, staining with HMB-45 was stronger in the unprotected halves compared with the protected halves (Figure). This is in concordance with the results reported by Tronnier et al.⁷

In summary, we extend the dermoscopic findings observed by Carrera et al² into the field of solar-simulated UV radiation, and we agree that not all UV-induced changes are confined to unprotected areas. Additional studies have to be conducted to elucidate this (unexpected) observation.

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Acquisition of data: Wolf, Gringschl, and Hofmann-Wellenhof.

Analysis and interpretation of data: Massone, Wolf, Gringschl, and Soyer.

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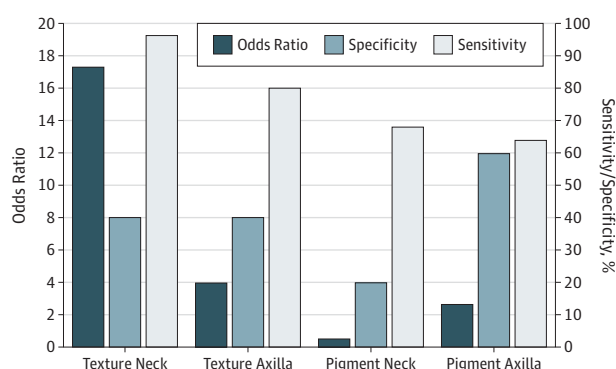
Posterolateral Neck Texture (Insulin Neck): Early Sign of Insulin Resistance

Acanthosis nigricans (AN) is so closely linked with insulin resistance (IR) that it has been called a clinical surrogate for laboratory-determined hyperinsulinemia.¹ The presence of AN can therefore indicate patients with IR, allowing implementation of interventions that may prevent progression to type 2 diabetes mellitus.² We report 30 patients who presented with elevated body mass index (BMI, calculated as weight in kilograms divided by height in meters squared) to correlate IR with different AN physical findings.

The homeostasis model assessment of insulin resistance (HOMA-IR) is widely used as an index of IR based on serum fasting glucose and insulin values: $\text{HOMA-IR} = [\text{glucose (mg/dL)} \times \text{insulin (}\mu\text{U/mL)}] / 405$. Esteghamati et al³ determined that IR is present in nondiabetic individuals if HOMA-IR is greater than 1.775.

Methods | Patients presenting to a private dermatology practice between September 2010 and February 2012 with BMI of at least 25 and with acrochordons or signs of AN, specifically hyperpigmentation and/or hyperkeratosis of the neck and/or axilla, were asked to participate in the study. Digital photographs of the neck and axilla were acquired, along with a patient history containing age, sex, race, BMI (using preexamination height and weight measurements), and personal and familial history of adult-onset diabetes mellitus. Fasting serum glucose and insulin values were obtained by means of glucose testing at an external testing site. The presence or absence of visible posterolateral neck pigment and/or texture and visible axillary pigment and/or texture were subsequently assessed from the photographs by 2 observers (W.V.S. and R.K.R.). This protocol was approved by the Phelps County Regional Medical Center institutional review board (Rolla, Missouri), in accordance with the Belmont Report.

Figure 1. Physical Findings for Acanthosis Nigricans



Neck texture has higher sensitivity and odds ratio for the homeostasis model assessment of insulin resistance than neck pigment or axillary texture and pigment. Sensitivity indicates percent of insulin resistance with acanthosis nigricans finding. Specificity indicates percent of non-insulin resistance without acanthosis nigricans finding.